

Peritoneal Papillary Serous Carcinoma: Study of 15 Cases and Comparison With Stage III–IV Ovarian Papillary Serous Carcinoma

BENJAMIN PIURA, MD, FRCOG,^{1*} MIHAI MEIROVITZ, MD,¹ MARY BARTFELD, MD,¹
ILANA YANAI-INBAR, MD,² AND YORAM COHEN, MD³

¹Unit of Gynecologic Oncology, Department of Obstetrics and Gynecology, Soroka Medical Center and Faculty of Health Sciences, Ben-Gurion University of the Negev, Beer-Sheva, Israel

²Institute of Pathology, Soroka Medical Center and Faculty of Health Sciences, Ben-Gurion University of the Negev, Beer-Sheva, Israel

³Institute of Oncology, Soroka Medical Center and Faculty of Health Sciences, Ben-Gurion University of the Negev, Beer-Sheva, Israel

Background and Objectives: Peritoneal papillary serous carcinoma (PPSC) is histologically and clinically similar to stage III–IV ovarian papillary serous carcinoma (OPSC). The purpose of this study was to investigate the clinical findings, treatment, and outcome of PPSC patients compared with stage III–IV OPSC patients.

Methods: Data from the files of 15 PPSC patients and 52 stage III–IV OPSC patients who were managed at the Soroka Medical Center between January 1991 and December 1997 were evaluated.

Results: With regard to patients' characteristics, presenting signs and symptoms, type and extent of surgery, tumor response to first-line chemotherapy, recurrence-free interval, recurrence site, tumor response to second-line chemotherapy, and serum CA-125 levels, no significant differences were observed between the PPSC patients and the stage III–IV OPSC controls. The prevailing presenting symptoms were abdominal mass and ascites. The mainstay of treatment was debulking surgery followed by adjuvant platinum-containing chemotherapy. The objective response rate to first-line chemotherapy was 80%. The actuarial 5-year survival rate for the PPSC patients and stage III–IV OPSC patients was 52.0% and 20.5%, respectively ($0.05 < P < 0.1$).

Conclusions: The clinical and surgical characteristics of patients with PPSC are similar to those of patients with stage III–IV OPSC. When treatment strategies for stage III–IV OPSC are applied to PPSC, the survival of PPSC patients may be similar or even better than that of stage III–IV OPSC patients. *J. Surg. Oncol.* 1998;68:173–178. © 1998 Wiley-Liss, Inc.

KEY WORDS: papillary serous carcinoma; debulking surgery; staging; chemotherapy; survival

INTRODUCTION

The Müllerian papillary serous carcinomas form a spectrum of tumors composed of peritoneal papillary serous carcinoma (PPSC), ovarian papillary serous carci-

*Correspondence to: Benjamin Piura, MD, FRCOG, Department of Obstetrics and Gynecology, Soroka Medical Center and Faculty of Health Sciences, Ben-Gurion University of the Negev, P.O. Box 151, Beer-Sheva 84101, Israel. Fax No.: (972) 7-6403503.
E-mail: piura@bgumail.bgu.ac.il

Accepted 10 April 1998

noma (OPSC), and uterine papillary serous carcinoma (UPSC). These tumors, despite the differences in their site of origin, have similar histologic and clinical features. OPSC is the most common and best recognized Müllerian papillary serous carcinoma, whereas PPSC is a relatively uncommon tumor that accounts for 7–21% of all epithelial ovarian carcinomas [1–5]. The adoption of the International Federation of Gynecology and Obstetrics (FIGO) staging of epithelial ovarian carcinoma for use in PPSC has presented a problem, since from the start PPSC is an intra-abdominal disease that must be regarded as at least stage III. Thus, when a study is designed to compare PPSC patients with OPSC controls, the OPSC controls should be patients with stage III–IV disease. Management of PPSC has followed that of epithelial ovarian carcinoma with initial debulking surgery (total abdominal hysterectomy, bilateral salpingo-oophorectomy, omentectomy, and extirpation of all resectable tumor masses) followed by adjuvant platinum-containing chemotherapy as the mainstay of treatment.

The Soroka Medical Center (SMC) in Beer-Sheva is the only tertiary care medical facility in the south of Israel that provides hospital care for a population of 500,000: Jews from various ethnic origins make up 80% of the population and Arab-Bedouins make up the remaining 20%. The aim of the present study was to assess the clinical and histologic findings, treatment, and outcome of 15 patients with PPSC compared with 52 patients with stage III–IV OPSC managed at the SMC over a 7-year period.

MATERIALS AND METHODS

The clinical and pathological records of 15 PPSC patients and 52 stage III–IV OPSC patients who were managed between January 1991 and December 1997 at SMC, Beer-Sheva, Israel, were reviewed.

The pathologic diagnosis of PPSC was based on the following Gynecologic Oncology Group's (GOG's) inclusionary criteria for PPSC [6]: (1) both ovaries must be either physiologically normal in size or enlarged by a benign process; (2) the involvement in the extraovarian sites must be greater than the involvement on the surface of either ovary; (3) microscopically, the ovarian component must be one of the following: (a) nonexistent, (b) confined to ovarian surface epithelium with no evidence of cortical invasion, (c) involving ovarian surface epithelium and underlying cortical stroma but with any given tumor size less than 5×5 mm, (d) tumor less than 5×5 mm within ovarian substance associated with or without surface disease; and (4) the histological and cytological characteristics of the tumor must be predominantly of the serous type that is similar or identical to OPSC, any grade.

Treatment for both PPSC and OPSC usually included initial surgical debulking followed by adjuvant systemic

chemotherapy. Sometimes, if the patient was considered not feasible for initial surgery, she had an interval surgery after a few cycles of neoadjuvant chemotherapy. Surgical debulking and staging usually consisted of peritoneal washings or collection of ascites if present, scrapings of the undersurfaces of the diaphragm, resection of tumor masses, total abdominal hysterectomy, bilateral salpingo-oophorectomy, omentectomy, peritoneal and serosal biopsies, and sampling of paraaortic and pelvic lymph nodes. In all patients at initial laparotomy, an attempt was made to debulk the tumor load as much as possible. Surgical debulking was considered "optimal" if at the end of surgery the largest residual tumor mass left in the abdominal cavity was less than 1 cm in its largest diameter. Although there is no official surgical staging system for PPSC, all cases of PPSC were considered to be the equivalent of FIGO stage III or IV ovarian cancer.

After surgery, patients were generally treated with intravenous platinum-based combination chemotherapy. The most prevailing chemotherapy regimen for both PPSC and OPSC consisted of cisplatin 75 mg/m² and cyclophosphamide 750 mg/m² at 3-week intervals. None of the patients received radiotherapy. Postoperatively, during and after treatment with chemotherapy, the patients were monitored with serial determinations of serum CA-125, and periodic ultrasound and computerized tomographic examinations. None of the patients had second-look laparotomy. Recurrent disease was documented in patients in whom serum CA-125 levels returned to normal and who were free of disease after initial surgery and then, during or after first-line chemotherapy, demonstrated rising levels of serum CA-125 and/or developed evidence of recurrent tumor in either the abdomen and/or pelvis or elsewhere.

The PPSC patients were compared to the OPSC patients with regard to age at initial diagnosis, menstrual history, parity, ethnic origin, past medical history, use of hormone replacement therapy, family history of cancer, presenting signs and symptoms, type and extent of surgery, stage of disease, type and response to first-line chemotherapy, recurrence-free interval, recurrence sites, type and response to second-line chemotherapy, serum CA-125 levels, and results of follow-up.

Evaluation of statistical significance of the difference between means was performed by the Student *t*-test [7]. Difference in the frequency of observations was evaluated by the χ^2 test and in a 4-fold table by the χ^2 test with Yates correction (χ^2_y) for small numbers [7]. Survival was calculated using the Kaplan-Meier method [8] and compared statistically using the log-rank test [9].

RESULTS

The clinical characteristics of the two groups of patients are detailed in Table I. The differences between the

TABLE I. Clinical Characteristics of PPSC and OPSC Patients*

Characteristics	PPSC (n = 15)	OPSC (n = 52)
Mean age (years) at		
Diagnosis ^a	62.0	55.6
Menarche ^b	12.3	11.0
Menopause ^c	50.6	48.0
Mean No. of children ^d	2.4	2.7
Ethnic origin ^e		
Ashkenazi Jewish	9 (60.0%)	38 (73.1%)
Sephardic Jewish	5 (33.3%)	13 (25.0%)
Arab-Bedouin	1 (6.7%)	—
Unknown	—	1 (1.9%)
Use of HRT ^f		
Estrogen + progesterone	1 (6.7%)	3 (5.8%)
Estrogen alone	1 (6.7%)	4 (7.7%)
None	13 (86.7%)	45 (86.5%)

*HRT = hormone replacement therapy; PPSC, peritoneal papillary serous carcinoma; OPSC, ovarian papillary serous carcinoma.

^a $t = 0.538$; DF = 65; $0.5 < P < 0.6$ [not significant (NS)].

^b $t = 1.137$; DF = 65; $0.2 < P < 0.3$ (NS).

^c $t = 0.436$; DF = 65; $0.6 < P < 0.7$ (NS).

^d $t = 1.000$; DF = 65; $0.3 < P < 0.4$ (NS).

^e $\chi^2 = 6.788$; DF = 3; $0.05 < P < 0.1$ (NS).

^f $\chi^2 = 0.1968$; DF = 1; $0.5 < P < 0.75$ (NS).

PPSC and OPSC patients with regard to mean age at diagnosis, menarche and menopause, parity, and ethnic origin were not significant. The ovaries and/or uterus had not been previously removed in any of the 15 PPSC patients, whereas in 5/52 (9.6%) OPSC patients the uterus had been previously removed. Two of 15 (13.3%) PPSC patients and 7/52 (13.5%) OPSC patients had received estrogen replacement therapy. None of the patients in the two groups had either a metachronously or synchronously second primary malignancy. A family history of cancer was obtained in 2/15 (13.3%) PPSC patients (1 breast cancer and 1 endometrial carcinoma) and 11/52 (21.2%) OPSC patients (3 breast cancer, 3 ovarian carcinoma, 2 gastric cancer, 1 endometrial carcinoma, 1 uterine cervix carcinoma, and 1 liver carcinoma).

The presenting signs and symptoms are displayed in Table II. In both groups, the most prevailing presenting signs and symptoms were abdominal mass and ascites.

The surgical characteristics of the two groups of patients are detailed in Table III. With regard to type and extent of surgery, the differences between the PPSC and OPSC patients were not statistically significant. In the PPSC group, the ovaries were involved with tumor in 12/15 (80%) patients and were tumor-free in 3/15 (20%) patients. The FIGO staging of the tumor in the two groups of patients is presented in Table IV.

Primary chemotherapy and the patients' responses to primary chemotherapy are described in Table V. The differences between the PPSC and OPSC groups with regard to type of first-line chemotherapy, number of cycles of chemotherapy, and response to cisplatin-based

TABLE II. Presenting Signs and Symptoms of PPSC and OPSC Patients*

Sign/symptom	PPSC (n = 15)	OPSC (n = 52)
Abdominal mass ^a	15 (100.0%)	41 (78.8%)
Ascites ^b	9 (60.0%)	20 (38.5%)
Pleural effusion ^c	2 (13.3%)	6 (11.5%)
Nausea ^d	1 (6.7%)	5 (9.6%)
Vomiting ^e	1 (6.7%)	5 (9.6%)
Constipation ^f	2 (13.3%)	2 (3.8%)

*Some patients presented with a combination of signs and symptoms; therefore, percentage adds up to >100%.

^a $\chi^2 = 2.4112$; DF = 1; $0.1 < P < 0.25$ [not significant (NS)].

^b $\chi^2 = 1.3938$; DF = 1; $0.1 < P < 0.25$ (NS).

^c $\chi^2 = 0.0692$; DF = 1; $0.75 < P < 0.9$ (NS).

^d $\chi^2 = 0.7491$; DF = 1; $0.25 < P < 0.5$ (NS).

^e $\chi^2 = 0.7491$; DF = 1; $0.25 < P < 0.5$ (NS).

^f $\chi^2 = 0.5591$; DF = 1; $0.25 < P < 0.5$ (NS).

TABLE III. Surgical Characteristics of PPSC and OPSC Patients

Characteristics	PPSC (n = 15)	OPSC (n = 52)
Type of surgery ^a		
Primary	13 (86.7%)	44 (8.6%)
Interval	2 (13.3%)	8 (15.4%)
Extent of surgery ^b		
Optimal	10 (66.7%)	27 (51.9%)
Non-optimal	3 (20.0%)	17 (32.7%)
Palliative	2 (13.3%)	8 (15.4%)

^a $\chi^2 = 0.0461$; DF = 1; $0.75 < P < 0.9$ [not significant (NS)].

^b $\chi^2 = 1.1190$; DF = 2; $0.5 < P < 0.75$ (NS).

TABLE IV. FIGO Staging of PPSC and OPSC Patients*

FIGO stage	PPSC (n = 15)	OPSC (n = 52)
IIIB	—	2 (3.8%)
IIIC	12 (80.0%)	39 (75.0%)
IV	3 (20.0%)	11 (21.2%)

* $\chi^2 = 0.625$; DF = 2; $0.5 < P < 0.75$ (not significant).

chemotherapy were not statistically significant. Objective response to first-line cisplatin-containing chemotherapy was observed in 12/15 (80%) PPSC and 41/52 (78.8%) OPSC patients, whereas 3/15 (20%) PPSC and 11/52 (21.2%) OPSC patients were refractory to cisplatin-containing chemotherapy. Of the patients who enjoyed a complete response to primary therapy, a recurrence-free interval of more than 6 months was observed in 3/11 (27.3%) PPSC and 16/31 (51.6%) OPSC patients ($\chi^2 = 3.0484$; DF = 1; $0.05 < P < 0.1$). The most common recurrence sites in both groups were the abdomen and pelvis. Second-line chemotherapy utilizing agents such as cisplatin, carboplatin, paclitaxel, etoposide (VP-16), cyclophosphamide, and hexamethylmelamine was employed in 10/15 (66.7%) PPSC and 23/52 (44.2%) OPSC

TABLE V. Type of First-Line Chemotherapy, Number of Cycles, and Response to First-Line Chemotherapy in PPSC and OPSC Patients*

First-line chemotherapy	PPSC (n = 15)	OPSC (n = 52)
Type ^a		
CAP	—	4 (7.7%)
CP	11 (73.3%)	41 (78.8%)
TP	4 (26.7%)	7 (13.5%)
Mean No. of cycles ^b	7.5	7.0
Response ^c		
Complete response	11 (73.3%)	31 (59.6%)
Partial response	1 (6.7%)	10 (19.2%)
Stable disease	1 (6.7%)	—
Progress of disease	2 (13.3%)	11 (21.2%)

*CAP = Cyclophosphamide, doxorubicin, and cisplatin; CP = cyclophosphamide and cisplatin; TP = paclitaxel and cisplatin.

^a $\chi^2 = 2.45$; DF = 2; $0.25 < P < 0.5$ [not significant (NS)].

^b $t = 0.520$; DF = 65; $0.6 < P < 0.7$ (NS).

^c $\chi^2 = 5.3772$; DF = 3; $0.1 < P < 0.25$ (NS).

patients ($\chi^2y = 1.5328$; DF = 1; $0.1 < P < 0.25$). Objective response to second-line chemotherapy was observed in 2/10 (20%) PPSC and 8/23 (34.8%) OPSC patients ($\chi^2y = 1.5908$; DF = 1; $0.1 < P < 0.25$).

Serum CA-125 at the time of diagnosis ranged from zero to 4,078 U/ml (mean: 827.5 U/ml) and from zero to 3,896 U/ml (mean: 462.8 U/ml) in the PPSC and OPSC groups, respectively ($t = 1.066$; DF = 65; $0.2 < P < 0.3$). At the completion of first-line chemotherapy it ranged from zero to 50 U/ml (mean: 13.3 U/ml) and from zero to 4,522 U/ml (mean: 146 U/ml) in the PPSC and OPSC groups, respectively ($t = 1.066$; DF = 65; $0.1 < P < 0.2$).

Follow-up of the PPSC patients ranged from 1 to 77 months, with 8/15 (53.3%) patients followed for at least 5 years or until time of death. Follow-up of the OPSC patients ranged from 1 to 105 months, with 36/52 (69.2%) patients followed for at least 5 years or until time of death. Of the PPSC patients, 3/15 (20%) were alive free of disease, 7/15 (46.7%) were alive with disease, and 5/15 (33.3%) had died of disease. Of the OPSC patients, 11/52 (21.2%) were alive free of disease, 6/52 (11.5%) were alive with disease, 1/52 (1.9%) had died of intercurrent disease, and 34/52 (65.4%) had died of disease. The difference between the two groups in the proportion of patients who either were alive with disease or had died of disease was not statistically significant [12/15 (80%) PPSC patients vs. 40/52 (76.9%) OPSC patients; $\chi^2y = 0.01$; DF = 1; $0.975 < P < 1.0$]. However, the difference between the two groups in the proportion of patients who were alive with disease was statistically significant [7/15 (46.7%) PPSC patients vs. 6/52 (11.5%) OPSC patients; $\chi^2y = 7.077$; DF = 1; $0.001 < P < 0.01$]. Overall, the actuarial 5-year survival rate for the PPSC patients was 52.0% and that for the stage III–IV OPSC patients was 20.5% ($0.05 < P < 0.1$) (Fig. 1).

DISCUSSION

PPSC was first described by Swerdlow [10] as malignant mesothelioma in 1959. Very soon it has become apparent that with regard to histology, immunohistochemistry, cellular ultrastructure, epidemiology, and clinical behavior, PPSC is not distinguishable from OPSC and therefore should not be considered a malignant mesothelioma but a variant of OPSC [3–5,11,12]. Some authors [13] have not even considered PPSC as a different clinical entity from OPSC and therefore have not reported it separately from stage III–IV OPSC, whereas others [3,14,15] have considered PPSC to be a different clinical entity from OPSC and have reported it separately from OPSC.

We, like others [6,12], could not demonstrate significant differences between PPSC and stage III–IV OPSC patients with regard to patients' characteristics, presenting signs and symptoms, type and extent of surgery, tumor response to first-line chemotherapy, recurrence-free interval, recurrence site, tumor response to second-line chemotherapy, and serum CA-125 level. Like others [12], we have observed that the rate of successful debulking and the result of postoperative aggressive treatment with platinum-based combination chemotherapy were the same in both groups. Some authors [16], however, have reported a lower rate of optimal cytoreduction and decreased response to platinum-based chemotherapy in the PPSC group. The literature is conflicting regarding the relative survival in these two patient groups. Some authors [5,13,16] have reported a poorer survival for patients with PPSC compared to patients with OPSC, whereas others [17,18] could not find a significant difference in the survival between patients with PPSC and patients with OPSC. We have observed a better 5-year survival rate for the PPSC patients (52%) compared to the stage III–IV OPSC patients (20.5%), but the difference is only of borderline significance ($P < 0.1$). Moreover, we have noticed that although almost the same proportion of patients in each group either were alive with disease or had died of disease (80% vs. 76.9%, respectively), a significantly greater proportion of PPSC patients (46.7%) compared to OPSC patients (11.5%) were alive with disease. Thus, in this series, it seems that the PPSCs progressed more slowly and caused death over a more extended period of time than did the OPSCs.

The finding that the presently reported 15 PPSCs accounted for 22.4% of all stage III–IV intra-abdominal Müllerian papillary serous carcinomas seen during the study period corroborates previous studies that demonstrated that PPSC account for about one-fifth of all intra-abdominal Müllerian papillary serous carcinomas [1–5]. Although Arab-Bedouins make up 20% of the population in the south of Israel, we have observed that only 1/15 (6.7%) PPSC patients and none of the OPSC patients was

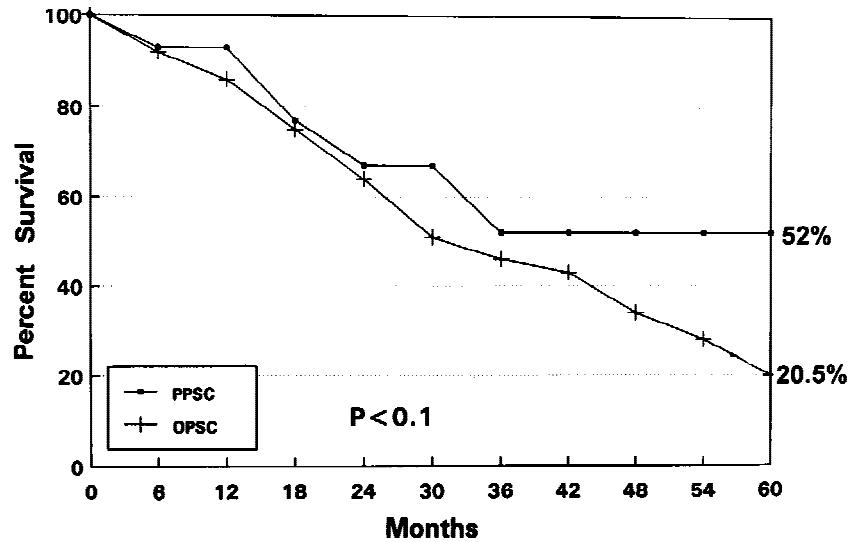


Fig. 1. Actuarial survival of PPSC and OPSC patients.

an Arab-Bedouin woman. Although Jews of Asian-African origin (Sephardic) make up 60% of the Jewish population in the south of Israel, we have noticed more women of European-American origin (Ashkenazi) than those of Asian-African origin (Sephardic) affected by PPSC and OPSC.

Like others [1,2,12,19], we have observed that the mean age at diagnosis of the PPSC and OPSC patients was about 60 years. The prevailing presenting symptoms of both PPSC and OPSC patients were abdominal mass and ascites. The finding in this study that 20% of the PPSC patients presented with stage IV disease does not exactly corroborate previous studies that demonstrated a greater proportion (28–32%) of PPSC patients with stage IV disease [2,11,16].

In contrast to some authors [20] who have shown that the ovaries were free of tumor in 7–14% of PPSC patients, we have found that the ovaries were free of tumor in 20% of the PPSC patients. Sometimes, because of extensive confluent spread of the tumor, it is difficult to identify during surgery and even on pathological examination whether the tumor distribution is consistent with extraovarian (PPSC) or ovarian (OPSC) origin. In this series, however, we did not encounter such a case.

Obviously, prophylactic bilateral oophorectomy prevents the development of OPSC, but cannot prevent the development of PPSC [21,22]. This raises doubts about the value of prophylactic oophorectomy at routine hysterectomy and in patients with familial ovarian cancer syndrome in totally preventing intra-abdominal Müllerian carcinomatosis [11,12,22]. Clinicians should explain to patients undergoing bilateral oophorectomy that although the risk of developing intra-abdominal Müllerian carcinomatosis is reduced (to approximately one-fifth of what it would have been had the ovaries been retained),

it is not totally abolished since papillary serous carcinoma may arise de novo from the peritoneal surfaces. In this series, however, none of the patients with PPSC had a previous bilateral oophorectomy.

In conclusion, this study indicates that with regard to patients' clinical and surgical characteristics, PPSC is similar to stage III–IV OPSC. It has been observed that the survival of PPSC patients was better than that of stage III–IV OPSC patients, but the difference is of borderline significance ($P < 0.1$). Nevertheless, the overall survival is low, but it is not unexpected in view of the advanced stage of disease.

REFERENCES

1. Fowler JM, Nieberg RK, Schooler TA, et al.: Peritoneal adenocarcinoma (serous) of Müllerian type: A subgroup of women presenting with peritoneal carcinomatosis. *Int J Gynecol Cancer* 1994;4:43–51.
2. Altaras MM, Aviram R, Cohen I, et al.: Primary peritoneal papillary serous adenocarcinoma: Clinical and management aspects. *Gynecol Oncol* 1991;40:230–236.
3. Fromm GL, Gershenson DM, Silva EG: Papillary serous carcinoma of the peritoneum. *Obstet Gynecol* 1990;75:89–95.
4. Feuer GA, Shevchuk M, Calanog A: Normal-sized ovary carcinoma syndrome. *Obstet Gynecol* 1989;73:786–792.
5. Mills SE, Andersen WA, Fechner RE, et al.: Serous surface papillary carcinoma. A clinicopathologic study of 10 cases and comparison with stage III–IV ovarian serous carcinoma. *Am J Surg Pathol* 1988;12:827–834.
6. Bloss JD, Liao SY, Buller RE, et al.: Extraovarian peritoneal serous papillary carcinoma: A case-control retrospective comparison to papillary adenocarcinoma of the ovary. *Gynecol Oncol* 1993;50:347–351.
7. Swinscow TDV: "Statistics at Square One." London: British Medical Association, 1983.
8. Kaplan EL, Meier P: Non-parametric estimation from incomplete observations. *J Am Stat Assoc* 1958;53:457–481.
9. Peto R, Pike MC, Armitage P, et al.: Design and analysis of randomized clinical trials requiring prolonged observation of each patient. II. Analysis and examples. *Br J Cancer* 1977;35:1–39.
10. Swerdlow M: Mesothelioma of the pelvic peritoneum resembling

- papillary cystadenocarcinoma of the ovary: Case report. *Am J Obstet Gynecol* 1959;77:197–200.
11. Dalrymple JC, Bannatyne P, Russell P, et al.: Extraovarian peritoneal serous papillary carcinoma. A clinicopathologic study of 31 cases. *Cancer* 1989;64:110–115.
12. Ben-Baruch G, Sivan E, Moran O, et al.: Primary peritoneal serous papillary carcinoma: A study of 25 cases and comparison with stage III–IV ovarian papillary serous carcinoma. *Gynecol Oncol* 1996;60:393–396.
13. Gooneratne S, Sassone M, Blaustein A, et al.: Serous surface papillary carcinoma of the ovary: A clinicopathologic study of 16 cases. *Int J Gynecol Pathol* 1982;1:258–269.
14. Strnad CM, Grosh WW, Baxter J, et al.: Peritoneal carcinomatosis of unknown primary site in women. A distinctive subset of adenocarcinoma. *Ann Intern Med* 1989;111:213–217.
15. Fox H: Primary neoplasia of the female peritoneum. *Histopathology* 1993;23:103–110.
16. Killackey MA, Davis AR: Papillary serous carcinoma of the peritoneal surface: Matched-case comparison with papillary serous ovarian carcinoma. *Gynecol Oncol* 1993;51:171–174.
17. Chen KT, Flam MS: Peritoneal papillary serous carcinoma with long-term survival. *Cancer* 1986;58:1371–1373.
18. Lele SB, Piver MS, Matharu J, et al.: Peritoneal papillary carcinoma. *Gynecol Oncol* 1988;31:315–320.
19. Ransom DT, Patel SR, Keeney GL, et al.: Papillary serous carcinoma of the peritoneum. A review of 33 cases treated with platinum-based chemotherapy. *Cancer* 1990;66:1091–1094.
20. Russell P, Bannatyne PM, Solomon HJ, et al.: Multifocal tumorigenesis in the upper genital tract. Implications for staging and management. *Int J Gynecol Pathol* 1985;4:192–210.
21. Chen KT, Schooley JL, Flam MS: Peritoneal carcinomatosis after prophylactic oophorectomy in familial ovarian cancer syndrome. *Obstet Gynecol* 1985;66(Suppl):93S–94S.
22. Tobacman JK, Greene MH, Tucker MA, et al.: Intra-abdominal carcinomatosis after prophylactic oophorectomy in ovarian cancer-prone families. *Lancet* 1982;2:795–797.